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NUTRITION REVIEWS

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NUTRITION REVIEWS

VOL. 4 DECEMBER, 1946 No. 12

NUTRITIONAL ASPECTS OF THE CANCER PROBLEM

Cancer is not ordinarily thought of as a disease in which diet is particularly important, and most of the emphasis of investigators has been placed on such aspects as genetics, hormonal influences, viruses, and exposure to specific toxic agents. Nevertheless, since the behavior of living forms can be modified by nutrition in practically all phases of the life cycle, it is only natural that the question should have been raised whether the development of abnormal tissue might also be modified by diet. Such has proved to be the case.

One of the more striking observations in the field of cancer research is that the formation of certain neoplasms can be influenced by the kind or amount of food ingested. Claims to this effect are not new, but it was only after suitable advances had been made in the science of nutrition as a whole, that reproducible dietary experiments with tumor-bearing animals became possible.

The development of a tumor, whether in man or in experimental animals, can be pictured as consisting of at least two stages: the stage during which the cancer cell arises as such, and the stage during which a sufficiently large aggregate of tumor cells is present to grow autonomously without requiring the further presence of any obvious carcinogenic factor within the animal body. A third "intermediate" or "critical" stage has likewise been postulated (B. E. Kline and H. P. Rusch, *Cancer Research* **4,** *762 (1944)*).

Experience both with human cancers and with tumors of experimental animals has indicated that it is relatively difficult to modify the progress of a tumor once it has been formed, except by the use of relatively deleterious agents such as radiant energy, or compounds related to mustard gas. Nevertheless, it is possible to retard the formation of tumors in experimental animals by restricting the caloric intake of the animal by 30 to 40 per cent of the amount that would ordinarily be consumed ad libitum (A. Tannenbaum, *Cancer Research* **2,** *460 (1942)*; H. P. Rusch, *Physiol. Rev.* **24,** *177 (1944)*). Such restricted animals usually lose weight for a while and then stabilize their weights at a somewhat lower level than that of fully fed animals. The rate of development of tumors in such animals is largely suppressed, and in some cases is suppressed entirely. The effects of dietary restriction are more marked upon tumor formation than upon the rest of the animal, and these effects have been noted with various types of tumors. This is significant since cancer is not a single disease but should rather be regarded as a family of diseases, depending upon which tissue is involved and upon the characteristics of the various types of tumors that are possible. Unfortunately, however, the effects of caloric restriction upon the formation of tumors are not permanent, and when a full intake of calories is resumed, the growth of the tumor begins anew.

A reduced caloric intake appears to be more effective as an anticarcinogenic influence during the period in which cancer cells arise. This has been demonstrated with many different types of tumors including those due to ultraviolet light, to specific carcinogenic hydrocarbons applied in different ways, and in spontaneous tumors that arise in purebred strains of mice. The appearance of mammary tumors, for example, can be prevented completely in susceptible strains of mice by a diet adequate in all essentials, but suboptimal in calories—"suboptimal" that is, so far as growth and maximum body weight are

concerned, but "optimal" for prolonging the life of the individual. That these results may have some bearing upon the formation of human cancer is suggested by insurance statistics on the relation of body weight to cancer incidence. The proportion of persons who die from cancer (all types) is greatest among those classified as "overweight" at the time their policies were issued; and it is somewhat greater for those of average weight than those considered underweight (A. Tannenbaum, *Arch. Path.* **30,** *509* (*1940*)). These results may be somewhat disconcerting to enthusiasts for more and better nutrition, since the criteria of a good diet usually involve a maximum rate of growth and heavier individuals during childhood. Nevertheless, the results are in harmony with the frequently quoted results of C. M. McCay, G. Sperling, and L. L. Barnes (*Arch. Biochem.* **2,** *469* (*1943*)) that underfeeding not only prolongs the life of rats, but also prolongs certain of the characteristics of youth (in the rat).

Indeed, experiments on tumor-bearing animals often suggest that "improvements" in the diet, such as the addition of more of the B-vitamins, more protein, or a better mineral balance, "improve" the rate of development and growth of the tumor, and hence hasten the death of the individual.

Results which are more in line with the hopes of nutritionists are those dealing with factors that modify the development of liver tumors in rats fed azo dyes. The appearance of such tumors can be delayed markedly by feeding extra amounts of riboflavin, egg white, or certain saturated fatty acids found in hydrogenated coconut oil (H. P. Rusch, C. A. Baumann, J. A. Miller, and B. E. Kline, *A. A. A. S. Research Conference on Cancer, p. 267* (*1945*)). Conversely, one of the more effective ways of producing this type of tumor rapidly is to feed a diet in which the source of the vitamin B-complex is a rice bran concentrate particularly low in riboflavin. Thus, it would appear that this type of tumor at least might almost be regarded as one aspect of a deficiency disease. Hepatic tumors have also been observed in experimental animals exposed for long periods of time to diets that favor the development of cirrhosis. These include diets containing selenium, or diets free from specific carcinogenic agents but high in fat, low in protein, and particularly low in labile methyl groups. These effects of diet on the formation of liver tumors in animals have an interesting parallel in man. Although primary carcinoma of the liver accounts for less than 1 per cent of all tumors among the people of the western nations, its incidence in Orientals and native Africans is high. In Japan, primary liver cancer ranks third in frequency of all cancer cases; it is equally common in other Asiatic countries, and the frequency among the South African natives is amazingly high. It accounted for 90.5 per cent of all cases of cancer among the Bantu Negroes recruited from the native territories or from Portuguese East Africa and who were employed in the Witwatersrand gold mines. Although parasitic infection, especially with liver flukes, has been mentioned as a possible cause of the high incidence of liver cancer in these people, the evidence points to diet as a major contributing factor. Cirrhosis of the liver is found in 4 out of 5 autopsies performed on male Bantus at the Johannesburg General Hospital (C. Gilbert and J. Gillman, *Science* **99,** *398* (*1944*)). This is perhaps directly related to dietary intake, since, because of poverty, these people are forced to live almost exclusively on the cheapest available carbohydrate, corn "mealie" meal. This food is partly cooked and in some areas it is supplemented by fermented cow's milk. Meat is a luxury and is eaten only on ceremonial occasions or when sheep or cattle die from disease or starvation. To ascertain the effect of the Bantu diet on the incidence of disease, mealie meal and fermented milk were fed to 125 albino rats for a period of

two years. Many signs of dietary deficiency were observed, and without exception the animals developed liver lesions which varied from a marked fatty change to multilobular cirrhosis. Frank cirrhosis occurred in 20 per cent. The similarity between the condition of the experimental animals and that of the underfed human beings was too striking to be ignored.

Nevertheless this does not mean that all cancer formation is due to a deficient diet, nor does it suggest that dietary constituents such as riboflavin, egg white, lauric acid, or "tergitol" should be employed as cancer cures. The latter factors retard only the formation of tumors of the liver. Once the neoplasms are large enough to be grossly detectable, their growth is not checked by feeding an abundance of any of these "inhibitors." Nor have these agents shown any appreciable effect on tumors arising in other organs. A diet even so poor as that of the Bantu is seldom carcinogenic *per se*. Tumor formation and subsequent growth are dependent on many factors, including the degree of autonomy of the neoplastic cell, and the effectiveness of defense mechanisms in the surrounding tissues. It is possible that neoplastic cells develop within normal tissues more frequently than is realized, but that few of these centers ever become established in the body. Hepatic tumors might be more frequent in peoples of Asia and South Africa, not because the diet induces cancer, but rather because it lacks certain factors essential to a defense mechanism. Unfortunately the formation of cancers for the most part does not appear to be due to inadequate diets; if this were the case, the elimination of neoplastic growths from the race would merely involve the discovery and administration of the missing factors, a large enough task to be sure, but very much more straightforward than the devious lines of experimentation now considered necessary in the study of cancer.

One hesitates to venture whether it will ever be possible to reduce the incidence of human cancer by dietary restriction. The food preferences and habits of people being what they are, any attempt to restrict total intake and yet retain a well balanced diet would be no easy matter even under proper supervision. Left to the individual himself, the result would almost certainly be an unhappy one since there would be no advantage in retarding one disease only to have others flourish. Perhaps at the present time the possibilities of dietary restriction could best be tested postoperatively in individuals who have had all visible cancers removed surgically. Caloric restriction might then serve to suppress the development of invisible metastases that have not been excised.

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PRESENT KNOWLEDGE OF THIAMINE IN NUTRITION (PART I)

Thiamine remains one of the substances of which the biologic activity for animals resides in a single entity. Although this entity may be free or combined in foodstuffs, another compound with vitamin B_1 activity has not been described. The heat lability of the vitamin was of importance in the identification of the factor. Modern investigations (*Nutrition Reviews* **2**, *143* (*1944*)) have confirmed that thiamine is

more stable to heat in lower pH ranges, but have demonstrated that at a given pH the stability is tremendously influenced by the presence of certain salts. There is evidence that foodstuffs may contain thiamine-stabilizing materials. No valid generally applicable estimate of thiamine loss in cooking can be made.

The most widely employed methods for determining this vitamin are some modifications of (1) the two chemical procedures involving oxidation of thiamine to thiochrome which can be measured by its fluorescence, or a colorimetric procedure based upon the Prebluda-McCollum reaction, which is a coupling reaction with a diazotized amine; (2) the yeast fermentation procedure of A. S. Schultz, L. Atkin, and C. N. Frey (*Ind. Eng. Chem. (Anal. Ed.)* **14,** *35 (1942)*); (3) microbiologic assays employing Phycomyces blakesleeanus or L. fermentum 36 (H. P. Sarett and V. H. Cheldelin, *J. Biol. Chem.* **155,** *153 (1944)*); or (4) biologic assays using rats, chicks, or pigeons as test animals. The many modifications of these various procedures which have been published indicate the general desire for more specific, dependable, and less tedious methods. Probably the most widely used method for clinical study is some modification of the thiochrome technic (*Nutrition Reviews* **4,** *148 (1946)*). Adaptations of this procedure which retain the adsorption step and which employ internal standards appear to be among the most reliable for studies of urinary excretion. Thiochrome procedures applied to urine usually give lower values than do yeast fermentation methods applied to the same urine.

Phosphorylated thiamine serves as the prosthetic group of a family of enzymes which D. E. Green terms thiaminoprotein enzymes (*"Mechanisms of Biological Oxidations," p. 120, Cambridge University Press, London (1941)*). These enzymes are concerned with the metabolism of alpha-keto acids, particularly pyruvic acid (E. S. G. Barron, C. M. Lyman, M. A. Lipton, and J. M. Goldinger, *J. Biol. Chem.* **141,** *957 (1941)*). D. E. Green, W. W. Westerfeld, B. Vennesland, and W. E. Knox (*Ibid.* **145,** *69 (1942)*) demonstrated the presence in pig heart, rabbit muscle, and other animal tissues of a thiamine-containing enzyme which catalyzed the decarboxylation of alpha-ketoglutarate, of pyruvic acid, and of alpha-ketobutyrate, while E. S. G. Barron, J. M. Goldinger, M. A. Lipton, and C. M. Lyman (*Ibid.* **141,** *975 (1941)*) showed that addition of phosphorylated thiamine to tissues of B_1-avitaminotic rats increased the utilization of alpha-ketoglutarate of the tissue. Thiamine is also involved in the oxidation of acetaldehyde, the first breakdown product of alcohol (*Nutrition Reviews* **3,** *356 (1945)*).

Pyrithiamine, a synthetic analogue of thiamine in which the —S— of the thiazole ring is replaced by —C=C—, possesses antithiamine properties for both microorganisms and for mice (*Nutrition Reviews* **2,** *59 (1944)*). The 2-n-butyl homologue of thiamine, differing from the latter only in that the n-butyl group replaces the methyl group attached to the pyrimidine nucleus, serves as a vitamin inhibitor for rats (*Ibid.* **4,** *110 (1946)*). The property of compounds to act as antivitamins is believed to depend upon structural relationships to the vitamin which enable the antivitamin to enter into one portion of some essential cellular reaction which is then unable to proceed. Thiamine, in sufficient concentration, reverses the effect of its two known antivitamins. Neither of these antivitamins is known to occur in nature. There is, however, in certain fish a heat-labile enzyme which destroys thiamine (*Ibid.* **1,** *61 (1942)*; **1,** *403 (1943)*; **2,** *128 (1944)*). Attention was directed to this thiamine-destroying enzyme (thiaminase) by the appearance of "Chastek's paralysis" in foxes fed a diet containing 10 per cent or more of uncooked fish. This disease is characterized by anorexia, weakness, progressive ataxia, spastic paraplegia, and hyperesthesia. The similarity of the focal lesions of the nervous system in this paralysis

in the fox and the lesions seen in Wernicke's syndrome in man have lent support to the concept that the latter is in part attributable to thiamine deficiency. Ingestion of raw clams with meals reduces the availability of thiamine for man by approximately 50 per cent (*Ibid.* **4,** *74* (*1946*)), presumably because of the presence of thiaminase in the clams. The importance of thiaminase in causing thiamine deficiency would depend upon the frequency with which raw fish were consumed. There is also evidence for the existence of a heat-stable thiamine inactivator in certain cereals, legumes, and oils. The nature and mode of action of this material are at present obscure (*Ibid.* **4,** *222* (*1946*)).

Thiamine no longer appears to exert a specific effect on the conversion of dietary carbohydrate into body fat, but rather the failure of thiamine deficient rats to synthesize and deposit fatty acids is due to a diminished food intake (G. E. Boxer and DeW. Stetten, *J. Biol. Chem.* **153,** *607* (*1944*)); *Nutrition Reviews* **1,** *58* (*1942*)). Likewise, a reinvestigation has shown that no specific defect occurs in tyrosine and phenylalanine metabolism in thiamine deficient rats (*Ibid.* **4,** *179* (*1946*)).

The past few years have seen little clarification of the gross physiology of thiamine in man. Most workers agree that the symptoms of both wet and dry beriberi are due largely to thiamine deficiency. Mild polyneuritis has been produced in 2 women maintained on a daily thiamine intake of 0.175 mg. per 1000 calories for a period of some four months (*Nutrition Reviews* **1,** *252* (*1943*)). On more severely restricted diets the polyneuritis did not appear. These observations are consistent with the effects of various levels of thiamine intakes in experiments with birds (*Ibid.* **1,** *28* (*1942*)), and with reports of thiamine deficiency in the cat and monkey (*Ibid.* **3,** *21* (*1945*)), and they should be interpreted properly. They signify that thiamine is concerned in the maintenance of a functionally normal nervous system. They should not be interpreted as indicating that all neuropathy, even of undernourished persons, is attributable to thiamine deficiency (*Ibid.* **4,** *56* (*1946*)). The neurologic disease known as Wernicke's syndrome is characterized clinically by clouding of the consciousness, ataxia, and ophthalmoplegia (paralysis of the ocular muscles), and pathologically by focal degeneration and hemorrhage in the brain. It is usually associated with alcoholism. The present evidence is consistent with the concept that this syndrome is associated with a disturbance of pyruvic acid metabolism and with thiamine deficiency, as well as with deficiencies of other dietary essentials (*Ibid.* **1,** *24* (*1942*)).

A definite relationship of thiamine deficiency to cardiac function has long been recognized. In rats this deficiency leads to a bradycardia, and this has been employed as an assay technic (A. N. Drury, L. J. Harris, and C. Maudsley, *Biochem. J.* **24,** *1632* (*1930*)). Acute thiamine deficiency in man may terminate fatally as cardiac failure (B. S. Platt and G. D. Lu, *Quart. J. Med.* **5,** *355* (*1936*)). Much has been written of the "beriberi heart" which is usually characterized as exhibiting enlargement, especially right-sided, decreased circulation time, elevated or normal venous pressure, less difference than normal between the oxygen content of arterial and venous blood, and a dramatic recovery following specific therapy. This concept has been confirmed (*Nutrition Reviews* **1,** *139* (*1943*)), including the decreased circulation time and the increased cardiac output. These latter aspects were explained on the basis of myocardial failure and an increased volume of the peripheral vascular bed due to dilatation of arterioles. Pronounced changes in the cardiac musculature such as focal or diffuse necrosis which, in some cases, was replaced by scar tissue, have been observed in thiamine deficient pigs (*Ibid.* **1,** *314* (*1943*)). Pigs exhibiting such lesions showed electrocardiographic

changes, but these were not pathognomonic (*Ibid.* **2,** *38* (*1944*)).

Most of the cases of "beriberi heart" reported in this country have been associated with alcoholism. A recent series of 12 cases has been reported from Cincinnati by M. A. Blankenhorn, C. F. Vilter, I. M. Scheinker, and R. S. Austin (*J. Am. Med. Assn.* **131,** *717* (*1946*)). These investigators suggest the following criteria for a diagnosis of "beriberi heart": (1) enlarged heart with normal sinoauricular rhythm, (2) dependent edema, (3) elevated venous pressure, (4) peripheral neuritis or pellagra, (5) nonspecific changes in the electrocardiogram, (6) lack of other recognized cause of heart failure, (7) clinical improvement with reduction of heart size after specific treatment, or autopsy findings consistent with beriberi (*Nutrition Reviews* **4,** *24* (*1946*)). In 11 of the 12 cases reported the response to thiamine was not dramatic. "It is thought that the duration and the severity of the deficiency play a part in the reversibility or irreversibility of the process." At present it is impossible to be certain of the role which thiamine plays in the causation of such cardiac conditions, and the relative roles of other deficiencies and of factors other than nutritional. Most clinicians regard the rapid response to specific therapy as the sine qua non of "beriberi heart."

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The Nutrition Foundation was organized by food and related manufacturers in December, 1941, as a sincere expression of their interest in scientific progress and human health. The basic purposes of the Foundation are: (1) the development of a comprehensive program of fundamental research, providing basic information in the science of nutrition; and (2) the support of educational measures that will assist in making the science of nutrition effective in the lives of present and future generations.

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